The Course of Childhood Schizophrenia

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Clinical disorders which are characterized by a prolonged and fluctuating time course provide particularly vexing problems for the evaluation of therapeutic results. Chance alone makes it inevitable that spontaneous remissions will coincide from time to time with almost any therapeutic measure if it be employed with sufficient frequency. When such chronic disorders occur in children, their complexity is further multiplied by the developmental process, with its own internal clock, which now races ahead, now slows down, and is likely to be deranged by the illness itself. Once again, it seems inescapable that steps in maturation will now and again follow the introduction of new treatment programs. The very eagerness of the investigator, as a physician, to relieve the suffering caused by the illness makes him all too ready to assign the changes observed to the agent he is administering at the time.

If there be any characteristic of childhood schizophrenia which is manifest in all clinical accounts, that characteristic lies in its long and fluctuating course. From the considerations advanced above, the puzzling phenomena of observed behavior can be regarded as the resultant of at least two interacting, irregularly oscillating determinants: (*a*) the somatopsychic or psychosomatic pathology, and (*b*) the developmental process, each reinforced or retarded by environmental factors. This very variability of the disorder sharply restricts the usefulness of individual case studies in the evaluation of treatment procedures. Each report, nevertheless, remains valuable, first, as an example of what can happen in childhood schizophrenia and, second, in providing clues as to possibly effective techniques in treatment. But to conclude from one or a few cases that method X *is* beneficial merely illustrates the prevalence of the *post hoc* fallacy.

The obvious answer—and the only final one—to the measurement of therapeutic effectiveness lies in a properly designed, controlled study with schizophrenic patients assigned to treated and untreated groups by careful matching or random placement. Such studies are, however, more readily conceived than executed. The condition is itself relatively uncommon, so that few centers can accumulate sufficiently large series. It is difficult to maintain an "untreated" group, for parents understandably seek relief for their child, not the answers to scientific curiosity, even if they admit that only such answers can ultimately solve the problem. Moreover, clinicians themselves seem too easily persuaded that a given method is the correct one if it be based upon a conceptual foundation to which they are committed. Others contend that psychiatric studies do not lend themselves to quantification, each case being different; consequently, they continue to rely upon intensive study of individual patients, some of whom improve, as indeed the remission rate tells us they must.

It will be revealing no secret to admit that, at this point, the ideal controlled study remains to be done. Are we then without guidelines in making prognostic judgment or in attempting at least a preliminary evaluation of the efficacy of suggested therapeutic measures?

There exist in the literature a number of reports of the vicissitudes of outcome in cases of childhood schizophrenia followed over fairly long periods of time. A review

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of the pertinent literature, in the light of the experience at the Children's Psychiatric Service of The Johns Hopkins Hospital, can provide at least the beginning of a "natural history" of schizophrenia in childhood, against which contentions of improvement ascribable to treatment can be measured.

Before we can attempt a critical evaluation of available follow-up studies, it is necessary to face the rather considerable problem provided by the wide variability in the conceptions of childhood schizophrenia and, consequently, in the criteria for diagnosis. The accumulation of reliable information about the course of a disease process and its modifiability by treatment presupposes comparability in the cases described as examples of the disorder. Could any significance be ascribed to a discourse on the treatment of "meningitis" if no attempt were made to cite species and type? It may be do not know in the former instance, as we argued that the analogy is inapplicable; we do in the latter, the etiologic agent or agents. But would we any longer discuss the prognosis of "mental deficiency" without distinguishing the many syndromes which we can differentiate on clinical grounds alone with as yet little or no clear idea as to their cause? The very success in distilling out recognizable entities has made most clinicians chary of generalizations about the still unclassified residuum of mental deficiency. Is not even greater caution indicated in the only lately charted field of childhood psychosis?

A brief historical note may serve to emphasize the recency of the renaissance of interest in childhood schizophrenia and the persisting need for careful description and analysis. Paradoxically, though the term *démence précoce* was coined in 1860 by Morel[1] for a psychosis in a 14-year-old boy, certainly as much child as adult, relatively little attention was given to the form of the earliest manifestations of dementia precox by subsequent students of the disorder. Kraepelin[2] is often cited as stating that onset occurred before the age of 10 in 3.5% of 1054 patients he reported. But on a closer analysis of his account we find him describing "a group of patients in whom already from childhood upwards a considerable degree of psychic weakness existed, although the more striking morbid phenomena only later, perhaps in the third decade, became noticeable and now led to fairly severe dementia."[3] He referred to these as cases of "engrafted" hebephrenia *(Pfropfhebephrenie)*, stressing the fact that "dementia praecox was in a certain manner grafted upon an already existing disease." Thus, we find that these cases were for the most part retrospectively diagnosed, and the differentiation between premorbid personality trends and the onset of psychosis per se was a matter of "uncertain and arbitrary"[3] judgment from the history supplied. Kraepelin appears to have had little actual experience with children as patients except for a number of cases of idiocy and imbecility, which he regarded as the end-state of an unrecognized dementia precox, on the basis of mannerisms and bizarre behavior. Bleuler,[4] with a more specific concept of the characteristics of schizophrenic psychopathology, stated in 1911:

> With relatively accurate case histories, one can trace back the illness to childhood, even to the first years of life, in at least five percent of cases. In this process, we completely disregard the anomalies which do not have a distinctly schizophrenic character.

The limitations of his experience with children are apparent in the further comment:

> At the present time, we know of no differences between the infantile and other forms of the disease . . . . The prognosis of those cases in which the onset of the illness occurs before puberty does not appear to be too poor for the next few years. What happens to them later I do not know . . . . However, the case histories of adults admitted to the hospital show that at least part of these early cases relapse and then usually become markedly deteriorated.[4]

Perhaps the first specific consideration of childhood schizophrenia as a separate category dates from the earliest papers (1905-1908) of de Sanctis, who suggested the term dementia praecocissima to designate this clinical subdivision. It became evident,

however, that de Sanctis included not only cases of childhood schizophrenia as most of us employ the term today, but as well others with chronic brain syndromes and severe mental deficiency.[5] Such scrap basket diagnoses, the loose usage of "childhood" to include onset as late as 16 or 17 years of age, and the predominant preoccupation of psychiatrists with hospitalized adults, each contributed to a growing disbelief in the reality of schizophrenia in childhood. As recently as 1942, Bender commented[6]:

> There are those who do not believe in childhood schizophrenia, not having seen a case. At the best, none of us has seen very many cases in which we could make a definite diagnosis, not knowing the acceptable criteria. There are others who, having seen certain types of mental disorders in children, prefer to call them schizophrenic-like psychoses of childhood.

No more than 10 years later we find Despert stating[7]:

> Although there are still people who believe that there is no such thing as schizophrenia in childhood, generally speaking we might say that the concept of childhood schizophrenia has passed from non-recognition to over-recognition.

Among the unconvinced minority remain such authors as Katan[8] who contend, on theoretical grounds, that schizophrenia cannot occur in childhood.

The contemporary era of interest can be said to date from the end of the third decade of this century. Early studies were far from convincing and were hotly contended. For example, in the same volume of the *American Journal of Psychiatry,* Burr,[9] then professor of mental diseases at the University of Pennsylvania, declared: "The youngest case of hebephrenic dementia praecox of which I have records occurred in a boy not quite 15 years of age," and Brill[10] described two patients, one 4½ and one 6 years, whom he considered catatonic. He referred to others, not elaborated upon, whom he had also seen. Critical scrutiny of Brill's capsule case histories, however, leaves considerable doubt as to the accuracy of the diagnosis of catatonia, even though one of these patients was later hospitalized at the age of 19 as a schizophrenic. More precise consideration of the problem of childhood schizophrenia began to emerge with such papers as that by Kasanin and Kaufman.[11] These authors, surveying admissions to the Boston Psychopathic Hospital from 1923-1925, found 21 of 65 children admitted with the diagnosis of schizophrenia. Of these 21, they considered 6 as "typical" schizophrenics, but the age of onset of several indicates a rather elastic concept of childhood. (For a more complete bibliography of early case reports, see Bradley[12] for the world literature up to 1941 and Goldfarb and Dorsen[13] for the literature in English through 1954.) Clarity began to emerge with Potter's[14] proposal of a general set of diagnostic criteria, which attempted to take into account the developmental level and intellectual idiosyncrasies of childhood as factors which necessitated a modification of classic notions of symptomatology. The first effort to describe clinical subdivisions among this heterogeneous group of disorders appeared with the contributions of Ssucharewa[15] and Grebelskaja-Albatz,[16,17] who proposed a division by mode of onset (acute or insidious) which was felt to have prognostic importance. Despert[18] later discriminated a third mode of onset: insidious with acute exacerbation, environmentally precipitated. Lutz, in a scholarly study of the world literature through 1936,[19,20] was able to locate some 60 cases that had been reported as instances of childhood schizophrenia. He took age 10 as the dividing line between childhood and adolescence as a stringent requirement to limit his consideration to prepubertal cases, a step which eliminated 30 cases. He reviewed the remaining reports by rigorous criteria in order that he might discern the distinguishing features presented by those children for whom the diagnosis was beyond question. The criteria employed were those of Bleuler and Homburger; the latter had stated that the most critical factor in making the diagnosis was the course of the disorder.[21] By these cautious standards, there remained only 14 cases in which one could speak with certainty of schizophrenia

(mit Sicherheit von einer Schizophrenie zu sprechen). He had, for example, dismissed the cases of de Sanctis, since many were clearly instances of organic brain syndromes *(organische Gehirnstörungen)*; he accepted only Cases 4 and 5 of Potter,[14] though he considered Cases 1 and 2 as presumptive *(wahrscheinlich)*. (The remaining cases were compiled from 3 by Weichbrod, 2 each from Vogt and Schnabel, and 1 each by Higier, Grebelskaja-Albatz, Tramer, Strohmayer, and Meier.) He added six cases of his own, which are reported in detail. He then summarized the observations on the series of 20 in terms of cause, frequency (less than 1%), course, manifestations, differential diagnosis, treatment, and prognosis: *(Die Entwicklung einer kindlichen Schizophrenie verläuft durchwegs ungünstig, ungünstiger als der Durchschnitt der Fälle bei Erwachsenen*—the development of childhood schizophrenia is always unfavorable, more unfavorable than in the average case among adults). His papers remain landmarks in the early literature on the subject.

The first major contribution to the differentiation of specific clinical types within the group of childhood schizophrenias was Kanner's delineation in 1943 of "early infantile autism."[22] He defined an entity whose pathognomonic features are extreme aloneness and an obsessive insistence on the preservation of sameness, with secondary symptoms in the spheres of communication and motor behavior.[23] Its onset, usually by the end of the first year of life and not later than the second, marks it as the earliest of the forms of schizophrenia.[23] The psychiatric literature from 1943 to 1948, while including additional contributions from Kanner,[25-26] is marked by a paucity of confirmatory papers by others, a finding which suggests reservation in accepting autism as an entity. Since 1949, however, the diagnosis has appeared with ever-increasing frequency[27-52]; the concept, now rather widely accepted, is being more and more widely applied, with a tendency toward dilution of its specificity.[23] It remains a classic example of the elucidation of a clinical entity through careful observation and analysis. It distills out of confusing heterogeneity a circumscribed syndrome, separable by symptoms and course from other psychotic states in children.

Kanner's pioneering effort was followed by Mahler's description of the "symbiotic infantile psychosis."[28,29] It is characterized by a later onset and distinguished by symptomatology centered about a desperate effort to avert the catastrophic anxiety of separation. Mahler and Settlage[53] have recently proposed a more elaborate classification with (1) early infantile autism, divided into (*a*) "resistive and negativistic" and (*b*) "pliable and passively conforming," and (2) symbiotic infantile psychosis, divided into the subtypes (*a*) "parasitic-clinging," (*b*) "searching for symbiosis," (*c*) "acute symbiotic," and (*d*) "secondary autism." Despite the theoretical clarity of this proposal, whether these subcategories will ultimately prove clinically useful remains a matter for experience to decide.

Meanwhile, Lauretta Bender, whose experience with childhood schizophrenia is by far the most extensive in this country, if not in the world, had been pursuing a different conception of the disorder. She had offered the formulation that it is "a clinical entity occurring in childhood before the age of 11 years which reveals pathology at every level and in every area of integration and patterning within the central nervous system, be it vegetative, motor, perceptual, intellectual, emotional, or social."[6] The emphasis is biological; the fundamental pathologic process is stated to be a diffuse encephalopathy[54] for which no confirming anatomical evidence is described. Bender has currently suggested a classification into clinical types: (1) the pseudodefective or autistic regressive type; (2) the pseudoneurotic or phobic, obsessive-compulsive, hypochondriac type, and (3) the pseudopsychopathic or paranoid, acting-out, aggressive antisocial type.[55] So large is the number of cases (850) reported

from the Bellevue group,[55] unparalleled in the experience of other clinics whose special interest in the problem makes them centers for referral of psychotic children, that inevitable questions arise as to whether the diagnostic concept is not so broad that many cases are included which others would not consider to be schizophrenic.

The recent period has seen other delineations of clinical types which may be regarded as falling within the framework of childhood schizophrenia, such as Bergman and Escalona's "children with unusual sensitivity to sensory stimulation" [56] and Ekstein's [57-59] "borderline" psychosis (also referred to as "schizophrenoid"). The wheel seems to have come full turn with the appearance of a concept that recalls de Sanctis' dementia praecocissima, but even more broadly conceived. Rank has introduced the notion of the "atypical child," by which "we refer to more severe disturbances in early development, which have been variously described as Heller's disease, childhood psychosis, childhood schizophrenia, autism, or mental defect." [60]

To include Heller's disease—with its termination in irreversible dementia and its histologically demonstrable cortical pathology [61]—and mental defect (!) in the same category with childhood schizophrenia—itself suspect of being a somewhat heterogeneous group of disorders—can serve only to confound confusion. It is as if we were to urge a return to "brain disease" as a category and include therein the multiplicity of neoplastic, vascular, toxic, and traumatic disease states which have in common only their predilection for nervous tissue; imagine, then, the futility of efforts to formulate prognosis or plan treatment. Yet this is precisely what the concept of atypical development proposes for psychotic states in children. Nor is Rank's point of view that of her group alone. Szurek,[63] reporting the consensus of his co-workers at Langley Porter clinic, states:

> We are coming more and more to the opinion that mild, moderate or even severe mental deficiency and organic brain disease can be complicated by severe mental disorder. . . . we are beginning to consider it clinically (that is, prognostically) fruitless, and even unnecesary, to draw any sharp dividing lines between a condition that one could consider psychoneurotic and another that one could call psychosis, autism, atypical development or schizophrenia.

There is no intention here of glossing over the very considerable problems in differential diagnosis.[64-65] Weygandt,[66] as long ago as the turn of the century, recognized the occurrence in patients with severe mental deficiency of symptoms paralleling those observable in schizophrenics. This, however, does not argue for the identity of diseases with overlapping symptomatology. The careful examination by Kallmann et al.[67] of genetic evidence for linkage between mental deficiency and schizophrenia by means of twin studies led them to the conclusion:

> Our findings definitely indicate that the endogenous forms of schizophrenia and mental deficiency are based on different factors which are specific and not related to each other.

Currently, there are a number of efforts to enhance differential diagnosis, such as to formulate more specific testing procedures those of Ritvo and Provence,[68] Provence,[69] and Bender and Helme.[70] Other experienced workers are attempting to describe the psychological core of the disorder; Rabinovitch [71] has introduced the concept of "dysidentity," which he considers the fundamental psychopathologic process; Despert [7] has offered the definition of childhood schizophrenia as "a disease process in which the loss of affective contact with reality, or failure to develop affective contact, is coincident with or determined by the appearance of autistic thinking and accompanied by phenomena of regression and dissociation." She sharply indicates the importance of differentiating schizophrenic from obsessive-compulsive states.[72]

At present, most workers would agree with Hirschberg and Bryant [64] when they point out: "In childhood schizophrenia, we are not dealing with a separate and distinct clinical entity; rather, we are dealing with a group of related and overlapping clinical syndromes." The task would seem to be one,

as Kanner [73] has worded it, of "a sober evaluation and integration of the knowledge so far attained and more knowledge yet to be gained. This will probably come best from an attempt to study and separate the syndromes which present themselves and from an assessment of the manner in which centrifugal and centripetal factors fuse and blend—or fail to fuse and blend—in each individual patient" (compare also Despert and Sherwin [97]).

This rather considerable, though necessarily incomplete, excursion into unresolved problems besetting the diagnosis of childhood schizophrenia serves to emphasize the major difficulty in the analysis of follow-up reports. Despite the lack of uniformity in criteria for diagnosis, most accounts fail to specify with precision the standards employed. Vagueness in this area is, unfortunately, further compounded by failure, in many instances, to indicate the meaning given to categories of improvement and nonimprovement. Nevertheless, despite these serious limitations, the available data provide at least a rough guide to the course of the various syndromes that comprise this group of disorders and a preliminary indication of their responsiveness to various therapeutic approaches.

Kraepelin, in his classic text, failed to mention any instances of recovery in the childhood form of dementia precox.[2] He considered two possible outcomes: one leading to severe idiocy; the other, to the more typical adult forms of the disorder. His evaluation of prognosis has, however, to be considered in the light of his conception of the basic process in this "disease," which, according to him, terminated inevitably in dementia; if it did not, the diagnosis had to be reconsidered. Bleuler's more meliorative note, already referred to,[4] that prepubertal cases do not appear to do too poorly "for the next few years," was based on limited opportunity for follow-up and is offset by his opinion that, when relapse occurs, childhood schizophrenics "usually become markedly deteriorated." Such childhood schizophrenics as were included in de Sanctis' dementia praecossima terminated for the most part in irreversible idiocy; once again, this seems implicit in the diagnosis itself.[5]

The study by Kasanin and Kaufman [11] included six cases considered by the authors to be "typical schizophrenia"; two, *Pfropfhebephrenie,* and five, "reactive psychosis." Of the first group, three made some degree of recovery; of the second, none, and of the third, all. However, all that was definite about the "recovered" cases was that they had been able to leave the hospital; their subsequent fate was unknown. Ssucharewa [15] reported 25 cases under 13 years of age, 20 with insidious and 5 with acute onset. All but three of the former group had normal I. Q. scores, but certain of the cases suggest organic syndromes. Outcome, unfortunately, is not clearly specified but appeared to have been uniformly poor for the period (?) of the study; the main distinction between the two groups lay in the more rapid deterioration of those with acute onset. Bradley described the conclusions of a later paper by Sukhareva (Ssucharewa) and Kogan [74] in the following terms: "None of their patients had a complete remission. The disorder left a slight defect in one-third of the children, moderate in one-third, severe in the remainder." Grebelskaja-Albatz reported 22 children schizophrenic by the age of 8 years. Of the nine cases with acute onset, all presented a fulminating course that terminated in mental retardation of varying degrees of severity.[16] Of the 13 cases with insidious onset, only one appeared to have recovered sufficiently to attend a normal school.[17] Length of the follow-up contact is uncertain. Lutz,[19] basing his evaluation on 14 cases in the literature and 6 in his own experience, commented:

Der Ausgang ist bei den bis heute beobachteten Fälle ausnahmlos ungünstig. Oft müssen diese Kinder als Pflegefälle in Irrenanstalten versorgt werden und leben dort als geistige Ruinen, bis eine interkurrente Krankheit ihrem Dasein ein Ende setzt. Es fehlen noch genaue Beobachtungen über den weiteren Verlauf solcher Psychosen. (The outcome in the thus far observed cases is without

exception unfavorable. Often these children must be cared for as custodial cases in mental hospitals and live there as spiritual ruins, until an intercurrent infection brings their existence to an end. We lack sufficient observations on the further course of such psychoses.)

Despert's paper,[18] the most extensive in the American literature of the 1930's, and a model of clarity, described the course of 29 children hospitalized at the New York State Psychiatric Institute between 1930 and 1937 and followed for one and one-half to six years. Nine were less than 7 years of age on admission, and twenty, between 7 and 13. Of the seven with acute onset, six "rapidly deteriorated" and one achieved "partial restitution" and "relative adaptation." Of the 16 cases with insidious onset, 3 rapidly deteriorated, 12 showed a chronic course with "ultimate lowering of the ideoaffective level," and 1, a chronic course with one exacerbation. Despert added a clinical description of a third type of onset: "insidious with an acute episode." Of the six children in this category, two achieved remission with adaptation, three displayed gradual lowering of the ideoaffective level, and one rapidly deteriorated. She regarded anxiety as a bad prognostic sign; of the 11 with this as a marked feature, 10 deteriorated rapidly. Despert stressed the psychopathologic significance of "dissociation between language sign and language function." In a subsequent review [75] of this material, Despert supplied important additional details on the age, prepsychotic personality, etc., of these children.

Lurie, Tietz, and Hertzman,[76] in analyzing the first 1000 cases at the Child Guidance Home, evaluated 20 as psychotic, 13 of whom were schizophrenic. There were eight cases with acute onset, three with acute onset on an insidious background, and two with insidious onset. Of the total group of 13, only 1 was able to make an adjustment in the community during the period of follow-up (1-13 years). Potter and Klein [77] reported the course of 14 schizophrenic children whose illness began between 4 and 12 years of age. Four showed some improvement at discharge, but three of these subsequently followed a downhill course. The authors concluded: "The outcome of the schizophrenic reaction group is exceedingly poor." Creak [78] reported 35 psychotic "children," of whom only 9 were 12 years or less at the time of onset. Of this latter group, the outcome was clearly poor in three, not specified in three but presumably poor in these as well, and good in three. Among the last cases, however, "Heather N." and "William B." were of a reactive character and "Basil L." questionably psychotic. Thus, in reviewing case reports through 1940, Bradley sadly concluded [12]: "The prognosis of childhood schizophrenia appears to be uniformly bad."

With the 1940's, however, there appeared a much more hopeful view of the prospects for these children. Cottington,[79] in describing β-erythroidin-modified pentylenetetrazol U. S. P. (Metrazol) shock treatment, maintained that some improvement was observable in six out of seven cases. The data presented, however, fail to support the conclusions in a study of extremely short duration. A second paper [80] on the same case material, enlarged to 15, reported 10 patients "definitely improved," of whom 7 showed "modification of behavior." The criteria employed for diagnosis were apparently Bender's, but those for evaluation of change were simply not specified by the author; the reader is left in the dark as to how to assess this work. A much more significant study was that by Lourie, Pacella, and Piotrowski,[81] who evaluated the cases of 20 children, schizophrenic by Potter's criteria,[14] ranging from 4 to 12 years of age at onset and followed for 4 to 11 years. Three of these cases, indistinguishable from the others initially, later developed signs of organic pathology and were consequently regarded as cases of symptomatic rather than true schizophrenia. Of the 17 "true" cases, 4 achieved an apparently normal adjustment in the community, scholastically as well as socially; 5 made a "fair to borderline" adjustment; 3 developed "typical adult schizo-

phrenia," and 5 failed to show any change or deteriorated. No clear relationship is apparent in the data between mode of onset and outcome, though acute onset early in life led to the worst result. The authors commented soberly:

> Recoveries or remissions showed no definite correlation with any type of treatment, direct or indirect, though the relation between direct psychotherapy or environmental change in individual cases seems evident. In half the cases found to be doing best in follow-up, recovery or remission was apparently spontaneous.

This investigation remains one of the few solid contributions available in the literature.

Despert [82] described her experience in outpatient psychotherapy with seven schizophrenic children; in the short period of her study (three months to two and one-half years) she noted decided improvement in three, slow progress in two, and uncertain or poor results in two. She concluded that outpatient therapy for such children may be of advantage because of the preservation of contacts with home (a conclusion directly at variance with that of Bettleheim,[83] who urged the importance of severance of the relationship between mother and child through residential treatment as the key to successful therapy). Chess and Rubin [84] reported nine children treated at a child guidance clinic and followed up to five years. The authors indicated that the project seemed worthy of further pursuit but made no claim of major gains through psychotherapy. In some of the cases cited, some argument is possible as to diagnostic allocation. Bender and Gurevitch [85] reported five young schizophrenic children treated with psychotherapy, three of whom also received electroconvulsive treatment. Of the five, four showed "distinct improvement" during the several years of the study. Szurek,[63] summarizing his clinic's psychotherapeutic experience with over 100 children (but one should recall his earlier comment about diagnostic specificity in this group), declared that 14 "can be counted on as well or as very markedly improved. They are now in school and have been living at home for several years and progressing rather well."

Williams and Freeman [86] evaluated 12 children they considered as schizophrenic (criteria unspecified) and "treated" with lobotomy. Two died postoperatively. Four were mute preoperatively; one of these acquired "a few words" after the procedure. Their main contention for this mutilating operation is that it reduces "destructive hyperkinesia"; not one of the patients achieved a normal life, though the task of nursing personnel was simplified. Sackler et al.[87] reported on 19 schizophrenic children (Bender's criteria), 13 of whom received histamine therapy. All of the cases had displayed "overt pathology" by the age of 4 years and were chronically ill. It was claimed that 12 out of 13 showed "improvement," but its nature and its duration are not clearly indicated.

The papers of the 1940's and early 1950's might be summarized in the following fashion: They are in agreement in finding an appreciable percentage of remissions among childhood schizophrenics, certainly as contrasted with the prevailing pessimism of the 1930's. Whether this reflects the results of earlier detection, better general treatment measures, or an extension of the diagnosis to cover cases of a milder nature, it is not possible to decide. Because of the small number of cases (5 to 30) followed in each of the reports in both eras, it is entirely possible that the differences are more apparent than real. The available information justified only the statement that remission could occur but did not permit a reliable estimate of its likelihood. More accurate prognostic judgment had to await the careful follow-up of larger series of cases; this task was dependent upon the accumulation of experience at those centers with long interest in the problem under the continuous direction of the same investigators; namely, Bellevue, under Lauretta Bender, and Johns Hopkins, under Leo Kanner.

The larger of the two major studies now in progress is that by Bender and her associates.[88] She has presented some preliminary results of an analysis of the subsequent

careers of 350 children diagnosed as schizophrenic between 1934 and 1946 and followed from a minimum of 5 to a maximum of 15 years later. There were 143 shock-treated patients (43 with Metrazol, 100 with ECT) and 50 children not so treated, who formed the first groups to be evaluated. Of the former 143, she was able to trace 120. We are told that 104 of these were seen at one time or another by other psychiatrists, who considered 69 of them to be schizophrenic (66%), 16 mentally deficient (15%), 10 to have personality disorders (10%), and 9 other psychoses (9%). When 26 of the 35 in the last three categories were reexamined by a Bellevue research staff, 22 were diagnosed as schizophrenic; 16 others, part of the original group of 120 but not examined elsewhere, were also found to be schizophrenic by Bellevue criteria. Thus, 107 of 120 childhood cases (89%) were diagnosed as schizophrenic by Bender's group 5 to 15 years after the original diagnosis had been made. This Bender considers confirmation of her concept of childhood schizophrenia.

We can agree with this contention insofar as the persistence of a schizophrenic behavior pattern into adolescence, when criteria for its recognition are presumably more widely accepted, attests to a continuum between the two forms of the disorder. On the other hand, unless we believe that once schizophrenic, always schizophrenic, we can conceive of remission or recovery without the persistence of a clinically detectable residuum. It is important to note, in contrasting diagnosis elsewhere (66%) with that at Bellevue (89%), that the criteria employed by Bender's group do not include a requirement that the patient be psychotic, and may be influenced by a prior diagnosis at their own hospital. This may account for the higher percentage of diagnostic "confirmation"; it also suggests the need for a cautious evaluation of the high percentage of schizophrenic progenitors (40%) reported among the families of their children. This finding, for example, contrasts sharply with out own [87] in the families of 100 autistic children (5%). Psychotic or neurotic states, in the Hopkins study, were explicitly restricted to disorders that led to hospitalization and/or psychiatric care.

As to outcome, Bender reported that two-thirds of the shock-treated group had required state hospital care after their stay at Bellevue, one-third continuously. At the time of the study, 50% were "in the community." "Twenty-five percent of the 50 now in the community are showing fair to good adjustment." (I presume "50" is meant to be "50%." It is later stated that "one fourth of the 143 shock-treated patients are making fair to good adjustment.") This result was contrasted with the outcome of 50 patients, not treated with shock, who were considered a "control" group. We are told that two-thirds were subsequently sent to state hospitals, one-third chronically, figures identical with those preceding; however, only two (4%) were making a "fair to good" adjustment. This difference is cited as an indication of the beneficial effect of ECT on prognosis. Unfortunately, the "control" group is largely (two-thirds) comprised of children whose parents refused treatment; the attitudes underlying this decision may very well be significant determinants in outcome. In view of the small number in the far from completely comparable "control" group and the great similarity of the course in other respects, the data supplied do not demonstrate any clear superiority for the treatment method.

One is left in further doubt as to the evidence for the claim for ECT in a later paper [55] that it results in "specific improvement in all but a minority" of treated childhood schizophrenics. If we examine, for example, the psychological studies of des Lauriers and Halpern [90] carried out on the very group of children whom Bender treated, we find these comments: ECT has "broken down all the superadded structure, the neurotic and anxious features, and there now appears in strong relief a clearly defined picture of schizophrenia. . . . There is no improvement in the mental continuum, there is no change in reasoning and judgment, the tendency to accept incidental and

far-fetched issues as important is still present . . . a general flattening of affect." The authors noted, somewhat wryly, that if ECT had done nothing else, it had served to clarify diagnosis, on post-, as opposed to pretreatment, testing. Other psychological studies at Bellevue [91] have demonstrated a lack of detrimental effects by ECT on intelligence, etc., but to show that it does not hurt hardly establishes the fact that it helps.

Moreover, Clardy [92] and Clardy and Rumpf [93] contend, with no less vigor, though with little more proof, that ECT has a bad effect and is dreaded by the patients themselves. Clardy reported 30 schizophrenic children, followed for 1 to 14 years. Group I (17 cases) differed from Group II (11 cases) in that the former had delusions and hallucination, but not the latter. Group III (two cases) differed from Groups I and II in manifesting less severe loss of contact. Of the first 17, 9 were "much improved" or "improved"; 8 of the second 11, and 1 of the last 2, at the time of the report. It is stated that the 10 who had received ECT became worse. The high percentage of favorable change noted stands in contrast with that in all the other papers in the literature. It is difficult to know just what is meant by the categories of improvement. Poor editing has resulted in manifest contradictions within the paper (I found eight unimproved, five improved, and four much improved in Group I, but elsewhere in the paper the figures are given as six, seven, and four; other errors can be noted). The second paper from Rockland State Hospital [93] described 32 patients "with schizophrenic manifestations," 30 of whom had received ECT at Bellevue. (Does this imply a diagnosis of schizophrenia there ?) The diagnosis was changed after "months of observation" to 20 cases of primary behavior disorder, 9 of childhood schizophrenia, and 3 of psychopathic personality. Of the schizophrenics, we are told that "all except two of this group of 11 [9 ?] had received electric shock treatment before admission to the children's unit. Before admission, the majority had been described as having shown initial improvement for several months and then as having relapsed or become worse." [96] We are informed that ultimately five were discharged, four as much improved, and were making a good adjustment to the community two years later. Eighteen of the "primary behavior disorders" were improved or much improved after psychotherapy; thirteen were doing very well one to two years later. Of the three patients with "psychopathic personality," one was much improved and one improved; the "much improved" patient was still in the hospital. The discussion, in referring to the previous report,[92] states that it included 10 (30 ?) cases, of which two-thirds made a good adjustment. Apart from the arithmetical inconsistencies in these two papers, they indicate a major dissent from the argument that ECT is of benefit in childhood schizophrenia, and at least imply a marked disagreement with the Bellevue criteria for its diagnosis. It is regrettable that the authors do not supply detailed and accurately reported data, which would be required in order to evaluate these contentions. Lack of proof for the value of ECT is, however, indicated in the last study we shall discuss.

The only study comparable in size to the Bellevue project is that carried out at Hopkins.[94] The population consisted of 80 autistic children who had been known to the clinic for at least four years and who had attained the age of 9 years or more. We were able to trace 63 (79%); the fragmentary data (two to three years) available on the lost cases were in line with the trends in the major group. The median and average age for the children studied was 15 years; the median and average follow-up period, nine years. Outcome was classified into three categories: "good" (patient functioning well academically and socially), "fair" (patient able to attend school at about grade level but distinctly deviant in personality), and "poor" (maladaptive functioning, characterized by apparent feeblemindedness and/or grossly disturbed psychotic behav-

ior). Of the total group of 63, 3 were classified as having a good, 14 a fair, and 46 a poor outcome. Thus, about 27% were functioning at a fair to good social level, a figure remarkable consistent with the Bellevue finding (25%).

On closer examination of the data, however, it became apparent that those children who failed to develop, or, once having developed, lost the ability to communicate by speech did much more poorly than the others. Taking as the line of demarcation the presence of speech with communicative value at the age of 5, we found 32 "speaking" and 31 "nonspeaking" children. Of the former group, 16 (50%) achieved a fair to good adjustment, whereas only 1 (3%) of the latter group did so. (The probability of this difference being due to chance alone is less than 1 in 1000.) Failure to develop speech may be regarded as an index of the severity of the autistic process; it, more than any other feature, seemed to determine outcome. So different is prognosis in the two clinical groups that the thought presents itself that we may be dealing with two syndromes rather than one. Against this is the observation that those originally speaking children with poor outcome were clinically similar to the bulk of the nonspeaking children in final appearance.

The peculiarities so characteristic of the autistic children continued to be apparent as time progressed. In our experience they have not developed in the direction of typical schizophrenic patterns with hallucinations and delusions. Their most salient features are the extreme degree of withdrawal and the obsessive traits. The almost total isolation from human relationships in the most severely autistic group dictates a progressive deterioration of intellectual functioning, so that many are now superficially similar to feebleminded children, though the more specific features usually remain evident to closer examination. The data supplied by this long period of observation tend to support the contention that early infantile autism is "the earliest possible manifestation of childhood schizophrenia." [23]

With respect to the question of possibly effective therapeutic measures, the study "failed to reveal any correlation between formal psychiatric treatment and the clinical outcome." [94] In our experience, ECT, various drugs, and intensive psychotherapy had no predictable effect on course. However, we were struck by the considerable efforts extended by schools and parents on behalf of those children who have improved; we cannot escape the feeling that these efforts were important in their recovery. Certainly, autistic children, if they have any potential for response, are in need of supportive measures which can reinforce their potentialities by creating the conditions for successful interpersonal relationships. We cannot, of course, predict with any certainty what vicissitudes of development the future holds for those children who are now functioning at a better level. Periodic reevaluation alone will supply the answers to the question of their ultimate adjustment in adult life.

Conclusions

At this point in the history of experience with childhood schizophrenia it would appear justified to state that about one-fourth of the cases can be expected to attain a moderately good social adjustment during adolescence, about one-third to deteriorate and require continuous institutionalization, and the remainder to fluctuate about a marginal level. From other considerations, the future prospects of 50%-75% of the total group of these children would appear to be rather poor; if they cannot function adequately under the relatively protected conditions of childhood and adolescence, it seems unlikely that they will survive the more stringent demands imposed by adulthood. These general statements can be made somewhat more definite in the group of autistic children; with adequate language function, about 50% have a chance of reaching a fair to good social outcome; in

its absence, almost none can be expected to achieve this level. There is as yet no evidence that any particular therapeutic agent (including electric shock therapy) can be depended upon to modify outcome of childhood schizophrenia, though the general measures of mental hygiene and "milieu therapy" should be pursued no less avidly in this group than in any other.

The data supplied by this survey of the literature provide a preliminary basis for comparison with claims of therapeutic efficacy. Unless the results of treatment programs indicate appreciably more than 25% of the cases showing substantial improvement, it will be difficult to conclude that the therapy has had a significant effect on outcome.

This review has also served to emphasize the lack of uniformity in criteria for diagnosis. This indicates, at the very least, the importance of specification of criteria in all future clinical reports. I have suggested, on another occasion,[95] that this urgent problem might possibly be in part resolved by organizing meetings at each of the leading centers and inviting key personnel from the other centers to the presentation of cases, considered typical and borderline in the estimation of the host clinic. Perhaps, out of free and full discussion there might emerge an operational definition of the group of childhood schizophrenias. The effort would still be worth while if it succeeded only in acquainting each clinician with the notions his colleagues employ. Until the etiology and pathogenesis of one or more of these syndromes can be scientifically established, such clinical efforts will be crucially necessary if we are to understand each other when we speak of childhood schizophrenia.

Harriet Lane Home for Children (5).

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